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Association of Change in Body Mass with Breast Cancer

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ABSTRACT

We examined the relation between maximal adult change in body mass and breast cancer in the Epidemiological Follow-up Study of the first National Health and Nutrition Examination Survey. A total of 5599 women ages 25 to 74 years at the baseline examination in 1971 to 1975 were analyzed. Adult body mass change was calculated from baseline interview questions on lowest and highest adult weights, ages at those weights, and adult height. The cohort was followed for a median of 10 years and yielded 101 cases of breast cancer.

In a multivariate model adjusting for potential confounders (age, body mass, education, parity, age at first birth, menopausal status, calorie and alcohol intake, and physical activity) the relative risk estimates for the upper two tertiles of body mass gain were 1.7 (95% confidence interval, 0.9 to 3.4) and 2.5 (95% confidence interval, 1.2 to 5.4), respectively, in comparison to the lowest tertile of adult body mass gain. The relative risk estimate for those with a loss in body mass during adulthood was 1.3 (95% confidence interval, 0.7 to 2.6) in comparison to those in the lowest tertile of adult body mass gain. There was no association between body mass at the baseline examination and subsequent breast cancer.

The results of this study suggest that gain in adult body mass is a predictor of breast cancer risk independent of adult body mass. These results also suggest that avoidance of marked weight gain during adult life may reduce the risk of breast cancer.

INTRODUCTION

Many (1–7), although not all (8–10), studies suggest that adiposity, or excess body fat, increases a woman's risk of postmenopausal breast cancer. Adiposity is thought to result in increases in bioavailable estrogen by a variety of mechanisms, including alterations in postmenopausal estrogen production (11), estrogen-protein binding (12), and 2- *versus* 16-hydroxylation of estradiol (13). The relation of adiposity to breast cancer is of considerable importance since adiposity is one of the few breast cancer risk factors that is potentially modifiable.

Most of the studies of the adiposity-breast cancer question have used body mass or weight as a surrogate measure for adiposity. Body mass or weight, however, may not accurately reflect adiposity. Two individuals with similar body mass or weight may have different lean body mass (fat-free mass, *i.e.*, muscle, bone, etc.) and, consequently, different degrees of adiposity. To the extent, then, that adiposity is a relevant factor in breast cancer etiology, the misclassification resulting from the use of these measures of adiposity would tend to diminish or obscure the adiposity-breast cancer association.

Since lean body mass decreases with age (14), adult weight gain largely reflects increased body fat and may be a better surrogate for adiposity than body mass indices. Analyses using body mass only during later adult life as a measure of adiposity will necessarily combine individuals with varying adiposity. The relation between adult weight gain and breast cancer has been extensively reported in only three studies to date. Each of these studies showed a direct association between adult weight gain and postmenopausal breast cancer (3, 4, 7). One of these studies observed no association between body mass and breast cancer (3). In the other studies the magnitude of the excess breast cancer risk was greater for adult weight gain than for increased body mass or weight (4, 7).

We report here the results of an analysis of the relation between change in adult body mass and subsequent breast cancer in a large cohort of women drawn from a sample of the United States population. Unlike prior workers we examine possible confounding by standard breast cancer factors, as well as alcohol and calorie intake and physical activity.

METHODS

The Cohort. The NHANES[2] I NHEFS was a prospective cohort study conducted in 1982–1984 by the National Center for Health Statistics and derived from NHANES I. Details of their design have been presented elsewhere (15, 16). The NHANES I surveys were conducted by the National Center for Health Statistics from 1971 to 1975 on a sample of the noninstitutionalized population of the United States. The surveys were designed to oversample population groups at high risk of malnutrition: children 1–5 years; women 20–44 years; the elderly ≥65 years; and low income individuals. These surveys provided cross-sectional information on demographic variables in this population. The overall NHEFS cohort consisted of 14,407 people ages 25–74 years at the time of their examination for NHANES I. Of the 8,596 women in this cohort, 83% were white. This total cohort included a total of 131 cases of breast cancer identified through hospital records or death certificates or both.

Population for Analysis. Women were excluded from the analytic cohort as follows: 675 of the original cohort could not be traced in 1981–1985; 483 women were found to be alive at that time but had no follow-up interview; and 12 women with a history of breast cancer on the first hospital record were excluded as representing prevalent cases. A total of 1710 (22 incident cases) women were excluded because they were missing complete baseline information on adult weight change. Other exclusions include 34 women whose weight was coded incorrectly and 83 women whose high and low weights were reported to occur in the same year. The final cohort comprised 5599 women, including 101 women who subsequently developed breast cancer.

Body Mass Change as Exposure Variable. Body mass change as an adult was derived from the following four questions at the baseline interview. "What is the most that you have ever weighed?" "How old were you then?" "What is the least you have weighed since you were 18?" "How old were you then?" Women who reported that their high weight occurred after their low weight were considered to have gained weight as adults. Women who reported that their high weight occurred before their low weight were considered to have lost weight as adults. Among women reporting weight gain, 91% reported low weight occuring under age 30 years. The majority (78%) of women gaining weight achieved their high weight in the decade prior to the baseline interview. Conversely, among women reporting weight loss, 72% reported high weight occurring under age 30 years. The majority (70%) of women losing weight experienced their low weight in the decade prior to the baseline interview.

Covariables. Information on the following potential breast cancer risk factors was obtained at the baseline interview: age; education; physical activity; weight; height; age at menarche; and age at menopause. Information on age at first birth and family history of breast cancer in the woman's mother or sister(s) was available only at follow-

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[2] The abbreviations used are: NHANES, National Health and Nutrition Examination Survey; NHEFS, National Health Epidemiological Follow-up Study.

up. The dietary data were derived from a 24-h recall interview conducted by a trained nutritionist using three-dimensional graduated food portion models. Information on smoking was collected at baseline on only 43% of the women in the original NHEFS cohort. Information on smoking at follow-up was used to infer baseline smoking status on the remainder of the cohort. Body mass indices were calculated as kg/m^2. Measured height at the initial examination was used to calculate body mass indices.

Statistical Analysis. The age-adjusted mean values of body mass gain and loss in subcategories of breast cancer risk factors and dietary variables were determined from least square means by the PROC GLM procedure in the SAS statistical package.

The influence of body mass change as an adult on breast cancer incidence was examined with age-adjusted incidence rates and with Cox's proportional hazards regression technique. Age-adjusted incidence rates were calculated by the direct method with the age distribution of the analytic cohort as the standard. Cox's proportional hazards regression was used to analyze the simultaneous effect of body mass change, age, and other factors on breast cancer incidence in the cohort. The analyses were performed with the PROC PHGLM procedure available in the SAS statistical package.

For the proportional hazards analyses, the group of women reporting weight gain was split into tertiles and the group of women reporting weight loss were assigned a distinct indicator. Women in the first tertile of weight gain served as the reference. Covariables were modeled as shown in Tables 2 and 3. Quartiles were based on the distribution of values for the entire analytical cohort. Among women gaining weight, a linear test for trend was carried out by modeling the body mass gain variable as a tertile trend variable (scored 0, 1, 2) in the proportional hazards analysis.

Proportional hazards models containing a multiplicative interaction term for body mass change crossed with each covariate (both modeled as trend variables) were run to test for effect modification between body mass change and each covariate. A larger model containing all of these interaction terms and the individual covariates was also run. The log likelihood ratio test comparing models with and without these interaction terms was used to test the statistical significance of these potential effect modifications.

RESULTS

The mean age of the analytic cohort at baseline was 48 ± 15 (SD) years. Fifty % of the women were under 45 years old and 25% were over 65 years old. Forty-three % of the cohort had not completed high school; 20% completed some education beyond high school. The mean follow-up time was 10 ± 2.0 years.

The distribution of body mass change is shown in Table 1. The range of body mass gain for the cohort was 0 to 48.0 kg/m^2; the median body mass gain was 7.2 kg/m^2. The range of body mass for the group losing weight was 0.4 to 34.7 kg/m^2; the median body mass loss was 5.8 kg/m^2.

The relation of both body mass gain and loss as an adult to possible breast cancer risk factors and to selected dietary and life-style variables is presented in Table 2. Both body mass gain and loss were greater in younger and less educated women, in women with higher parity, those with earlier age at menarche and first birth, and in postmenopausal women. Adult body mass gain and loss were also greater in women who were of higher body mass at baseline examination, were sedentary, consumed fewer calories, and were nondrinkers. Height, percentage of calories as fat, smoking habits, and family history of breast cancer were not associated with adult body mass gain or loss (data not shown).

Table 1 *Distribution of body mass (kg/m^2) change as an adult in NHANES cohort of 5599 women*

The median weight loss value is 33 pounds and the median weight gain values for the tertiles of body mass gain are 22, 40, and 70 pounds, respectively.

	Body mass loss group (n = 1343)	Tertiles of body mass gain		
		1 (n = 1419)	2 (n = 1418)	3 (n = 1419)
Mean	−6.4	3.7	7.2	13.2
Median	−5.8	3.9	7.1	12.1
Range	−0.4, −34.7	0, 5.0	5.1, 8.5	8.6, 48.0

Table 2 *Mean body mass change in relation to breast cancer risk factors and other life-style variables in NHANES cohort of 5599 women*

Variables	Mean body mass change[a] Loss (n = 1343)	Gain (n = 4256)
Age (yr)		
<45	6.7 ± 0.3[b]	8.4 ± 0.2
45–65	6.2 ± 0.3	8.3 ± 0.2
>65	6.1 ± 0.5	7.1 ± 0.3
Education (yr)		
<12	6.8 ± 0.2	8.8 ± 0.1
12	6.3 ± 0.2	7.8 ± 0.1
>12	5.8 ± 0.2	6.9 ± 0.2
BMI[c] (kg/m^2) at examination		
≤25.5	5.4 ± 0.1	5.6 ± 0.1
>25.5	7.4 ± 0.1	10.5 ± 0.1
Parity		
0	5.9 ± 0.2	7.0 ± 0.2
1–3	6.3 ± 0.1	7.8 ± 0.1
≥4	7.1 ± 0.2	9.0 ± 0.1
Age at first birth		
<20	7.0 ± 0.2	9.0 ± 0.1
20–24	6.4 ± 0.2	8.1 ± 0.1
25–29	5.6 ± 0.3	7.4 ± 0.2
≥30	5.9 ± 0.4	7.1 ± 0.3
Age at menarche		
≤12	7.6 ± 0.3	9.0 ± 0.2
13–14	6.3 ± 0.1	8.0 ± 0.1
≥15	5.5 ± 0.3	7.8 ± 0.2
Menopausal status		
Premenopausal	6.0 ± 0.2	7.8 ± 0.1
Postmenopausal	6.8 ± 0.2	8.4 ± 0.1
Total calories		
<1027	7.4 ± 0.2	9.0 ± 0.1
1028–1379	6.3 ± 0.2	8.2 ± 0.1
1380–1779	6.2 ± 0.2	7.6 ± 0.1
>1779	5.8 ± 0.2	7.5 ± 0.1
Alcohol		
None	6.6 ± 0.2	8.6 ± 0.1
Any	6.3 ± 0.2	7.5 ± 0.1
Activity		
Inactive	7.6 ± 0.3	9.8 ± 0.2
Moderately active	6.4 ± 0.2	8.2 ± 0.1
Very active	6.2 ± 0.1	7.6 ± 0.1

[a] Age-adjusted (age as a continuous variable) by least square means method, except for age groups.
[b] Mean ± SEM.
[c] BMI, body mass index.

Age-adjusted incidence rates and relative risk estimates for breast cancer from proportional hazards models for tertiles of body mass gain and for body mass loss are presented in Table 3. The age-adjusted relative risk estimate for women in the third tertile compared to women in the first tertile of body mass gain was 1.5 (95% confidence interval, 0.9 to 2.6).

Simultaneous inclusion of potential confounders resulted in increased relative risk estimates as seen in the multivariate model in Table 3. The relative risk estimate for women in the third tertile compared to women in the first tertile of body mass gain was 2.5 (95% confidence interval, 1.2 to 5.4). The risks increased in a stepwise fashion for increasing levels of body mass gain and the test for linear trend was significant (P =

Table 3 *Relative risk estimates and incidence rates for breast cancer by body mass change as an adult in NHANES cohort of 5599 women*

Body mass change groups	Cases/ cohort	Incidence rates[a] (per 100,000)	Relative risk estimates (95% confidence intervals) Age-adjusted[b]	Multivariate[c]
Loss	20/1343	160	1.0 (0.6–1.9)	1.3 (0.7–2.6)
Gain (tertiles)				
1	21/1419	160	1.0	1.0
2	25/1418	180	1.1 (0.6–2.0)	1.7 (0.9–3.4)
3	35/1419	220	1.5 (0.9–2.6)	2.5 (1.2–5.4)

[a] Age-adjusted by the direct method.
[b] Based on age-adjusted regression coefficients from the proportional hazards model; total of 101 cases. Among women gaining weight a test for trend of body mass gain modeled as a tertile trend variable yielded $P = 0.14$.
[c] Based on 90 cases with complete covariate information including age in years (<45, 45–65, >65), body mass index at examination (quartiles), education, parity, age at first birth, menopausal status at examination, calories, alcohol, activity (remainder of variables modeled as in Table 2). Among women gaining weight a test for trend of body mass gain modeled as above yielded $P = 0.03$.

0.03). The relative risk estimate for women who lost weight compared to women in the first tertile of body mass gain was 1.3 (0.7 to 2.6). There was no evidence of any statistically significant effect modification by any of the covariables.

DISCUSSION

In this cohort study of women from a sample of the United States population, an increase in body mass as an adult of over 8 kg/m^2 was associated with a 2.5-fold increase in breast cancer risk. This finding agrees with previous reports of increased adult weight gain in women with breast cancer compared to controls (3, 4, 7). In addition, the relative risk estimate for lesser body mass gain indicates a positive dose-response relationship between body mass gain and breast cancer.

It is possible that the positive association we observed between body mass gain and breast cancer reflects a peculiarity of the data rather than a true association. The large number of exclusions from the original NHANES I data set could have biased our findings. However, selection bias would explain our results only if the excluded group included a disproportionate number of women who were low weight gainers and who subsequently developed breast cancer. To assess this possibility, we examined weight gain from age 25 years to the follow-up examination in a subset of women from both the analytical cohort and the excluded group (those with no body mass index change data at the baseline examination; $n = 1827$) who developed breast cancer and had historical information on weight at age 25 years at the follow-up examination. There was no significant difference in body mass change from age 25 years to the follow-up examination among breast cancer cases from the analytical cohort and the excluded group, means of 5.2 and 3.7 kg/m^2, respectively, $P = 0.24$. We also examined the baseline body masses of women who were excluded. The mean body mass at the baseline examination for this group compared to that of the analytical cohort was identical, 25.6 kg/m^2. In addition, no differences were seen in any of the distributions of potential breast cancer risk factors between the analytical cohort and the original cohort.

We also considered that our choice of reference groups may have altered our results. The alternative group of women with stable weight, such as women with −10% to +10% weight gain contained only 239 women (including 4 cases) and, therefore, was too small to use as a reference group. We did not consider all women who lost weight to be an appropriate reference group because it was not possible to determine which women lost weight due to an associated disease.

It is possible that nonrandom errors in recall of past weight influenced our findings. Although recall of recent weight has been reported to be accurate (17, 18), there are no reports on the accuracy of recall of extremes of weight. Inasmuch as information on high and low weights was collected at baseline in this prospective study, it is unlikely that development of breast cancer biased recall of body weight in this cohort.

The substantial increase in the body mass gain relative risk estimates derived from the multiple risk factor compared to the age-adjusted model is noteworthy. Several protective breast cancer risk factors, such as high parity, early age at first birth and nondrinking status, were directly associated with body mass gain and loss. Although controlling for these factors individually had only small effects on the body mass gain estimates, the simultaneous inclusion of all the risk factors did substantially increase the relative risk associated with the third tertile of body mass gain (Table 4).

The previous studies that evaluated weight gain and breast cancer found increased adult weight gain in postmenopausal breast cancer cases compared to controls (3, 4, 7) but found no association (3, 7) or a decreased adult weight gain among premenopausal breast cancer cases (4). This seems plausible because excess body fat is thought to increase postmenopausal breast cancer risk by increasing bioavailable estrogen due to increases in extraovarian estrogen production (11, 19) and to changes in estrogen-protein binding (12). Prior to the menopause excess body fat is thought to have little influence on bioavailable estrogen due to the overriding influence of ovarian estrogen production (19).

In this cohort menopausal status at diagnosis is unknown for some women. Seventy-five precent of cases, however, were over age 50 years at the time of breast cancer diagnosis. In addition when we examined the subset of women who were postmenopausal at baseline, the multivariate relative risk estimates were 2.5 (0.9 to 6.5) and 3.9 (1.4 to 10.8) for the second and third tertiles, respectively, of body mass gain compared to the first tertile. The multivariate relative risk estimate for the group who lost weight compared to the first tertile of body mass gain was 1.6 (0.6 to 4.5). Therefore our findings are consistent with the above studies reporting increased adult weight gain in

Table 4 *Multivariate[a] relative risk estimates for breast cancer by body mass change as an adult in subgroups of the NHANES cohort of women*

Strata (cases/cohort)	Relative risk estimates (95% confidence intervals) for body mass change: Loss	Tertiles of gain		P^b
BMI[c] (kg/m^2)				
≤25.7 (48/2652)	1.3 (0.6–2.7)	1.6 (0.8–3.6)	3.4 (1.3–9.3)	0.02
>25.7 (42/2602)	2.1 (0.4–12.9)	2.5 (0.5–11.1)	3.8 (0.9–16.3)	0.04
Interval between high and low wt (yr)				
<20 (43/3244)	1.5 (0.6–3.5)	1.4 (0.6–3.6)	2.3 (0.8–6.8)	0.16
≥20 (47/2010)	0.8 (0.2–2.9)	2.2 (0.8–5.7)	3.2 (1.1–9.4)	0.06

[a] Models include variables as shown in Table 3 except for BMI in BMI-stratified models.
[b] Among women gaining weight a test for trend of body mass gain modeled as shown in Table 3.
[c] BMI, body mass index.

postmenopausal breast cancer cases compared to controls (3, 4, 7).

A previous analysis of body size and breast cancer in the NHANES I cohort found no association between body mass or weight at baseline and subsequent breast cancer (10). Similarly, in our smaller cohort, body mass at baseline (in models with and without body mass change) was not associated with breast cancer, whereas we found a significant increase in breast cancer risk associated with marked body mass change. This increase in breast cancer risk associated with marked body mass gain was greater among more obese women but was also present among leaner women as shown in Table 4. Compared to previously reported relative risk estimates associated with body mass or weight, the magnitude of the elevated risk associated with body mass gain in this cohort is greater. This finding is similar to previous reports (3, 4, 7) and suggests that adult body mass gain may be a more biologically relevant measure of adiposity and is a predictor of breast cancer risk independent of body mass.

Although the weight loss group in this cohort did not experience a reduced relative risk of breast cancer compared to the first tertile of body mass gain, we were unable to exclude women from this group who experienced weight loss due to underlying disease. Therefore, this weight loss group is not representative of healthy women with weight loss due to decreased intake or increased activity. In order to lessen the likelihood that the weight loss group included those with preclinical disease, we excluded the first 4 years of follow-up. However, this did not alter the relative risk estimates. It is possible that the interval of weight change influenced these estimates; therefore we also examined models stratified by the time interval between high and low weights as shown in Table 4. Among women who experienced a weight change over a prolonged interval (≥20 years) the relative risk estimate for women who lost weight compared to women in the first tertile of weight gain was 0.8 (0.2 to 2.9). The lack of an increased relative risk associated with weight loss over a prolonged period suggests that the increased relative risk among women with weight loss in the nonstratified model was due to the inclusion of women with shorter term weight loss. It is noteworthy that the relative risk estimates associated with weight gain over a similar prolonged interval were increased rather than decreased compared to the estimates from the nonstratified model, at 2.2 (0.8 to 5.7) and 3.2 (1.1 to 9.4), respectively, for the upper two tertiles compared to the lowest tertile of body mass gain.

The observation that excess weight gain increases breast cancer risk in postmenopausal women suggests that avoidance of weight gain might reduce risk in these obese women. Alterations in hormonal status associated with weight gain may explain part of such an effect. Recent studies in obese women have found decreased levels of sex hormone-binding globulin; this decrease is associated with increases in bioavailable estrogen (20, 21). In postmenopausal women with breast cancer, weight loss resulted in a return of sex hormone-binding globulin to the normal levels seen in nonobese women (21). Future studies examining changes in hormonal levels with weight loss in obese postmenopausal women without cancer may help to further define the role of weight gain and loss in breast cancer.

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